P057. Implantation model studies: integrins and adhesiveness of the apical cell pole of RL95-2, a human uterine epithelial cell line

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Introduction: At implantation, the initial contact between the human embryo and maternal tissues is by the adhesion of trophoblast to uterine surface epithelium. Uterine epithelial cells are normally repellent and do not allow other cells to adhere via the apical plasma membrane. At receptivity, however, uterine cells are functionally reprogrammed towards adhesiveness for trophoblast. Little is known about the molecular details of contact formation of this modified apical plasma membrane with trophoblast. Based on cycle-dependent changes in integrin patterns *in vivo*, we have designed experiments with uterine epithelial cell lines that may shed light on the role of integrins in human implantation.

Materials and methods: RL95-2 is a human uterine epithelial cell line expressing integrins in basal, lateral and apical plasma membrane domains. In addition, cells exhibit adhesive properties of the apical plasma membrane for trophoblast. RL cells were treated with cytochalasin D to affect localization of the integrins via elimination of the actin network. After treatment with cytochalasin D (0.4 μ M for 120 min), RL cells were examined for integrins (α_6 , β_1 , β_4) using immunocytochemistry. Adhesiveness of the treated cells was measured using a centrifugal force-based adhesion assay.

Results and discussion: We found that alteration of the actin filament system via cytochalasin D treatment significantly reduced the adhesive properties of RL cells for trophoblast. The decrease in adhesiveness was linked to changes in topography of the integrins (α_6 , β_1 , β_4). In contrast to untreated RL cells, the apical surface of treated cells was free of integrins, and molecules were clustered at the margins of cells. These results provide evidence that integrins may be involved in the adhesiveness of uterine epithelial cells when molecules are properly localized in the apical plasma membrane by cytoskeletal interactions. The particular arrangement of integrins in uterine epithelial cells may depend on a master gene programme triggered by steroids *in vivo*.



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